



NEET SS MEDICINE

**MEDICINE
FOUNDATION - 1**

MARROW

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Basics of Thyroid

★ 4.6 | 109 Min

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Graves' Disease

★ 4.8 | 89 Min

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Toxic Adenoma & Toxic MNG

★ 4.7 | 25 Min

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★ 4.8 | 22 Min

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★ 4.7 | 20 Min

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★ 4.7 | 54 Min

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Bone & Mineral Disease: Basics I

★ 4.7 | 27 Min

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Bone & Mineral Disease: Basics II

★ 4.8 | 33 Min

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**Parathyroid Hormone**

★ 4.7 | 33 Min

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**Hypercalcemia**

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**Hypercalcemia: Clinical Aspects**

★ 4.7 | 19 Min

https://www.youtube.com/watch?v=...

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**Hypercalcemia: Diagnosis & Management**

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★ 4.8 | 11 Min

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BASICS OF THYROID

----- Active space -----

Anatomy

00:01:40

Development :

- **First endocrine organ** to be formed.
 - Develops around **3 to 4 weeks**.
- It is an endodermal derivative :
 - It develops from the endodermal cells of the foregut.
 - Solid mass of endodermal epithelial cells proliferating from the floor of primitive pharynx.
 - Development occurs at the **thyroid pit/ analage** which is at the floor of primitive pharynx.
 - Primitive pharynx : Junction of the anterior $2/3^{\text{rd}}$ and posterior $1/3^{\text{rd}}$ of the tongue (m/c site for ectopic thyroid : Lingual).
 - The cells migrate via the thyroid diverticula.
- The thyroid diverticula migrates anterior to the trachea to form the thyroid gland :
 - By the end of **5th week** thyroid diverticula breaks down and atrophies completely.
 - The remnant of the diverticula in adults is called **foramen caecum**.
- Persistence of thyroid diverticula gives rise to thyroglossal cyst :
 - most common site : Sub-hyoid.
 - moves with protrusion of tongue.
 - Needs to be excised completely.
- Para-follicular cells/ C-cells :
 - It produces calcitonins :
 - The extend of spread of medullary cancer can be assessed by blood levels of calcitonin.
 - Differentiated medullary thyroid cancer produces **calcitonin**.
 - Undifferentiated medullary thyroid cancer produces CEA.
 - They develop from the **ultimobranchial body** (Derivative of neural crest and 5th pharyngeal pouch).

Size : 4 x 2.5 x 2.5 cm.

- When size is **>4cm** on USG, it is called thyromegaly.

----- Active space -----

Weight :

- 1-2 g at birth.
- 12-15 g in adulthood.
- 250 mcg of T4 per gram of thyroid tissue → Stored hormone in adult approximately **4000 mcg** (Good enough to survive for 7 weeks).

Blood supply :

Blood flows at the rate of **4-6 ml/min/g** (Good blood supply prevents infections).

Superior thyroid artery :

- Branch of external carotid artery.
- Closely related to external laryngeal nerve.
- Ligation done close to the gland to avoid injury to nerve.

Inferior thyroid artery :

- Branch of thyrocervical trunk, which is a branch of subclavian artery.
- It supplies the parathyroid gland also, so only capsular branches are ligated.

Lymphatic drainage :

- Central lymph nodes (Drains to first).
- Lateral deep cervical nodes :
 - upper pole of thyroid can directly drain into the lateral deep cervical nodes.
 - malignancy of **upper pole of thyroid** bypasses the central lymph nodes.

Hormone synthesis

00:12:44

Overview :

- Begins at 11 weeks.
- Earliest hormone to appear is thyroglobulin : **29 Days**.
- Thyroid binding globulin synthesis starts by 10 weeks.
- Thyroid hormone synthesis (T3 & T4) starts by **11 weeks**.
- TSH production : 14 weeks.
- Hypothalamopituitary axis is fully developed by **18-20 weeks**.

Thyroid hormones during intrauterine life (IUL) :

- Thyroid hormones are required for myelin formation and neurotransmitter function in a developing baby before 10th week of IUL → Obtained from **maternal T4** being transferred across the placenta :
 - maternal iodide deficiency → **Cretinism**.

----- Active space -----

- Neurological cretinism : maternal iodide deficiency + maternal thyroid hormone deficiency → No brain development → mental retardation/ spastic quadriparesis.
- If the deficiency persists through out the pregnancy → myxedematous cretin.
- maternal thyroid hormones :
 - T4, T3, TRH, thyroid related antibodies : Can cross the placenta.
 - TSH : Does not cross the placenta.
- β -hCG and TSH have same structure, so it binds to the TSH receptors on the thyroid gland to produce thyroid hormones (Specificity spill-over).
- Soon after birth there is TSH surge, which comes back to normal after 24 to 48 hours, after which TSH levels are checked using heel-prick method to rule out congenital hypothyroidism.
- Congenital hypothyroidism : Normal at birth with normal IQ.
 - If hormone replacement is started within 2 weeks of birth, child will be normal.

Congenital hypothyroidism vs cretinism :

Cretinism	Congenital hypothyroidism
Neurological & myxedematous features	Hypotonia, wide-open posterior fontanelle, macroglossia, umbilical hernia.
maternal iodine deficiency	Thyroid gland dysgenesis (80-85%) : Defect in transcription factors Dyshormonogenesis (15-20%) : TPO mutation (m/ c); transient congenital hypothyroidism

Thyrotoxicosis during pregnancy :

- During pregnancy (11th-14th week) some patients can have thyrotoxicosis symptoms :
 - H/o hyperemesis grvida/ vomiting.
 - This is due to β -hCG acting on the TSH receptors → Transient gestational thyrotoxicosis.
 - Usually self limiting, no treatment is required.
- Hydatidiform mole produces $\uparrow\uparrow$ β -hCG → Severe thyrotoxicosis symptoms.
- DDs for thyrotoxicosis during pregnancy :
 - a. Graves disease.
 - b. Transient gestational thyrotoxicosis.
 - c. Hydatidiform mole.

----- Active space -----

Transcription factors :

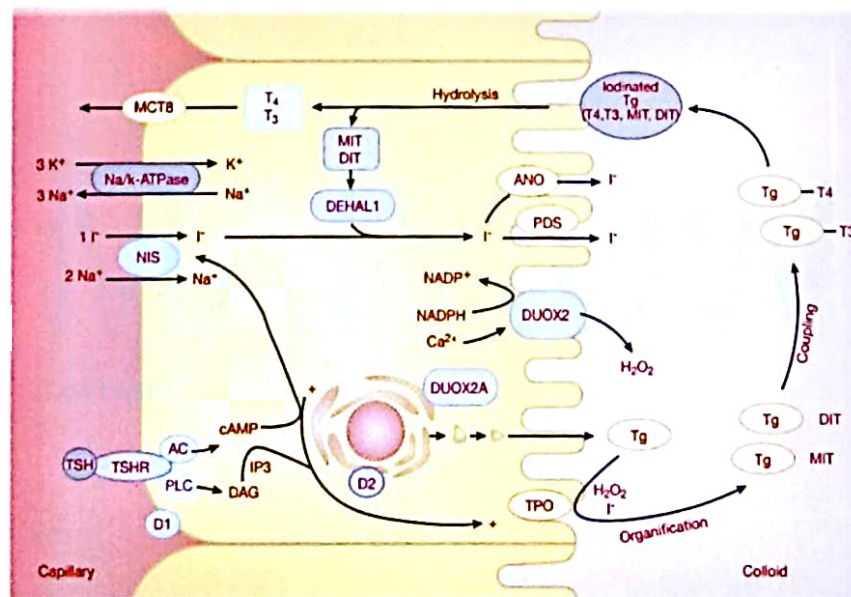
- Three most important factors include TTF-1 (NKX-2), TTF-2 & PAX-8.
- Defect of TTF-1/ NKX-2 is called **Brain-lung-thyroid syndrome** (Autosomal dominant):
 - Brain : Choreoathetosis.
 - Lung : Infant respiratory distress syndrome.
 - Thyroid : Thyroid dysgenesis.
- Defect in TTF-2 (FOX-E) (Autosomal recessive) :
 - **Cleft palate.**
 - Coanal atresia, spiky hair.
 - Thyroid dysgenesis.
- Defect in PAX-8 (Autosomal dominant) :
 - Renal agenesis.
 - Ureteral abnormalities.
 - Thyroid dysgenesis.
- **IGSF-1 & TBLX₁** : Cause **central hypothyroidism**.

Steps of thyroid hormone synthesis

00:31:36

Overview :

- 100% T₄ (84 mcg/day) & 20% T₃ (30 mcg/day) is produced from the thyroid gland.
- 80% T₃ is produced in the periphery from T₄.
- T₃ is clinically more important than T₄.
 - 3 Times more potent than T₄.
 - Free fraction of T₃ is 0.3% and T₄ is 0.02%.
 - Nuclear receptor binds only to T₃.
 - T_{1/2} of T₃ 18-14 hour & T_{1/2} of T₄ 1 week.



Thyroid hormone synthesis



Iodide transport across basolateral membrane :

----- Active space -----

- **Rate limiting step.**
- **Na-Iodide symporter :**
 - Iodide transport across basolateral membrane occurs via Na-I symporter.
 - It is also present in **salivary gland**, breast, placenta, GI mucosa, choroid plexus epithelial cell.
 - Thyroid scintigraphy/ thyroid radionuclide scan : Done with help of I^{123} & Tc^{99} .
- I^{123} is used for thyroid scintigraphy :
 - $T^{1/2}$ of 13 hours.
 - Works on the principle of being taken up via Na-I symporter and kept in the thyroid.
 - Normal uptake by thyroid gland is **10-15%** and is compared with the uptake in salivary gland.
 - Gamma rays are used.
 - Nowadays, I^{123} has been replaced by Tc^{99} as no organification is required for testing.
- Cold nodules have **low expression of Na-I symporter** hence have ↓ uptake.
- **Note :**
 - I^{124} : Used for PET scan.
 - I^{125} : Brachytherapy/ labelling agent for radio-immuno assay (RIA).
 - I^{127} : Naturally occurring form of iodide.
 - I^{131} : Radio iodine treatment ($T^{1/2}$ of 8 days).
- Na-I symporter is inhibited by 2 drugs :
 - Thiocyanate.
 - Perchlorates.
- At the end of this step, iodide is inside the follicular epithelial cells.

Iodide transport across the apical membrane :

- Done with help of **chloride-iodide antiport** (Pendrin/ Anoctamin).
- Pendred syndrome :
 - **SLC26A4 mutation** (Autosomal recessive).
 - Characterized by **goiter + sensory neural hearing loss**.
 - Pendrin gene is also seen in **cochlea** of the inner ear : malformation is called **mondini malformation of cochlea**. Patients are usually euthyroid; can have hypothyroidism.



----- Active space -----

Organification/ oxidation :

- Process of conversion of iodide to iodine ($I^- \rightarrow I_2$).
- Done with help of 2 enzymes :
 - Thyroid peroxidase (TPO) :
Heme containing enzyme.
TPO mutation is the m/c cause for dysmorphogenesis.
 - Dual oxidase (DUOX-2) :
NADPH containing enzyme.
Calcium requiring enzyme.

Iodination :

- Process of attachment of iodine molecules to tyrosine residues present over thyroglobulin molecule.
- End product : moniodo tyrosine (MIT)/ di-iodo tyrosine (DIT).
- This step requires TPO.

Coupling :

- MIT + DIT = T3.
- DIT + DIT = T4.
- Happens with the help of TPO.

Endocytosis :

- Thyroglobulin taken back into the follicular epithelial cells.

Release of T4 and T3 :

- Occurs via hydrolysis.
- Inhibited by iodides.
- Dehalogenase : Removes excess iodide and released back into the pool via pendrin.

Release of hormones :

- Requires receptor MCT-8 :
 - Only receptors that allows transport of T4 & T3 into the brain.
 - mutation of MCT-8 $\rightarrow$ Allan herndon dudley syndrome (AHDS) :
 - a. X-linked recessive.
 - b. Causes mental retardation and spastic paraparesis.
 - c. T3 toxicosis : T3 that had to be transported to the brain is channeled back into the blood.
 - d. TSH $\downarrow$, T4 $\downarrow$.